Sci TM Editor's Choice

Overline: MICROBIOLOGY

Title: Infections with benefits

One-sentence summary: Gastric *Helicobacter pylori* infection, which has paradoxical effects on human health, alters the immune environment and microbiota.

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Text of summary

Although the majority of the world's population carry a *Helicobacter pylori* infection without any symptoms, this bacterium has earned a bad reputation because of its causative association with stomach inflammation and peptic ulcer disease - a discovery that earned Marshall and Warren the 2005 Nobel Prize in Physiology and Medicine. However, accumulating evidence suggests that *H. pylori* can have unexpected health benefits, particularly in the young, including protection against asthma, certain types of infections, and esophageal diseases. In a recent issue of *Cell Reports*, Kienesberger *et al.* describe some of the details of the complex host response to *H. pylori* colonization.

The researchers established long-term, stable *H. pylori* infections in two cohorts of relatively young mice - one 4 weeks of age, just after weaning, and another at sexual maturity (6 weeks). Analysis of the mice at 1, 2, 3, and 6 months after infection showed that infected animals' stomachs had an inflammatory reaction, which was histologically very similar to that observed in humans. The mouse model also recapitulated the human endocrine response to *H. pylori* with elevated plasma concentrations of ghrelin, a versatile gut hormone that affects appetite, metabolism, and even the immune system. A comprehensive look at how the infection affected the expression of 547 genes related to the immune response in the stomach showed many changes, including persistent up-regulation of pro-inflammatory and T cell activation genes, as well as other temporally dynamic responses that may reveal clues about how the immune system adapts to persistent infection in parallel with ageing. Kienesberger and colleagues also described a changed composition of the microbiota "ecosystem," both in the stomach and downstream in the intestines of infected animals. This could be a cause or a consequence of the diverse host response.

A systemic manifestation of *H. pylori* infection in the stomach was implied by the change in ghrelin concentration, but by looking at the lung more closely, the authors determined that other organ systems are also affected. Analysis of immune cell populations in the lung revealed modestly more Th17 (pro-inflammatory T helper) cells, and a trend toward more regulatory T cells in infected mice. As for immune-related gene expression in the lung, there were only a few consistent changes, but early time-points showed up-regulation of many potentially influential genes, such as receptors for TGF-beta, IL4, and IL6.

It is interesting to question whether the systemic and generally immuno-suppressive response observed in this study is specific to *H. pylori*. It also remains to be seen whether this microorganism-triggered immune response, which likely evolved to facilitate bacterial survival, underpins the epidemiological link between *H. pylori* and reduced susceptibility to other conditions such as asthma.

Full citation, with authors and title

Kienesberger *et al*. Gastric *Helicobacter pylori* infection affects local and distant microbial populations and host responses. Cell Reports *14*, 1-13, February 16, 2016. Doi: 10.1016/j.celrep.2016.01.017

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